Cardiovasc Intervent Radiol (2014) 37:257–261
DOI 10.1007/s00270-013-0642-y

CASE REPORT

Critical Limb Ischemia in Association with Charcot Neuroarthropathy: Complex Endovascular Therapy for Limb Salvage

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Received: 10 March 2013 / Accepted: 3 April 2013 / Published online: 9 May 2013

Abstract Charcot neuroarthropathy is a low-incidence complication of diabetic foot and is associated with ankle and hind foot deformity. Patients who have not developed deep ulcers are managed with offloading and supportive bracing or orthopedic arthrodesis. In patients who have developed ulcers and severe ankle instability and deformity, below-the-knee amputation is often indicated, especially when deformity and cutaneous involvement result in osteomyelitis. Ischemic association has not been described but can be present as a part of peripheral arterial disease in the diabetic population. In this extreme and advanced stage of combined neuroischemic diabetic foot disease, revascularization strategies can support surgical and orthopedic therapy, thus preventing osteomyelitis and leading to limb and foot salvage.

Keywords Charcot foot · Critical limb ischemia · Endovascular therapy

Introduction

The incidence of Charcot neuroarthropathy in patients with diabetes is estimated to be 0.1–2.5 % [1]. This condition is a major cause of severe instability and is associated with a higher risk of amputation. Traditionally, patients who have not developed deep ulcers are treated by offloading and orthopedic arthrodesis. On the other hand, for patients who develop ulcers and osteomyelitis, the risk of amputation is high, and below-the-knee amputation is often indicated [2].

The severe joint instability causes the collapse of the tibia through the foot, with it reaching the ground during walking, and with a characterized protuberant fibular malleolus associated with a high risk of ulceration [3].

Charcot deformity in itself is not correlated with critical limb ischemia (CLI); nor has it been described in the current literature. However, during the natural history of diabetic foot syndrome, peripheral arterial disease could involve patients with Charcot foot.

Diabetic patients with a Charcot deformity associated with CLI have a major risk of ulceration and infection as a result of the combination of both risk factors, and orthopedic therapy alone, without revascularization, is often not successful for limb salvage.

Revascularization is the first-line treatment for CLI [4]. Endovascular therapy provides good clinical results and has been growing in acceptance as a primary therapeutic strategy for infrapopliteal and lesions of the foot arteries [5–9].

Here we present a case of ischemic Charcot foot deformity. The affected patient arrived with a foot ulcer and infection, then underwent combined endovascular, surgical, and orthopedic treatment for limb salvage.

Case Reports

An 83-year-old man with type II diabetes, hypertension, and Charcot foot deformity arrived for observation for forefoot phlegmon, Rutherford class 6, and a grade IIID lesion, as assessed by the Texas University classification (ischemic and infected ulcer with bone involvement) (Fig. 1A,B). At

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admission, the patient was diagnosed with CLI, with 8 mm Hg of transcutaneous oxygen tension ($TcPO_2$).

After surgical debridement of the lesion (Fig. 1C,D) and analysis of a previous angio-CT study to exclude lesions of the iliac arteries, the patient underwent endovascular treatment for below-the-knee lesions.

The patient had previously been treated with ticlopidine, 250 mg daily, and aspirin, 100 mg daily, 3 days before the procedure. The double antiaggregation was continued for 12 weeks after the procedure, and aspirin was prescribed for life. A total of 5,000 IU of heparin was administered intra-arterially at the start of the procedure.

Through antegrade access via the ipsilateral common femoral artery, performed under ultrasound guidance (Esaote 7.5 MHz, linear probe), and after administration of local anesthesia, the diagnostic angiographic studies confirmed the patency of the femoropopliteal axis, occlusion of the posterior and anterior tibial arteries, patency of the tibioperoneal trunk and peroneal artery, and, on the foot, occlusion of the pedal artery (Fig. 2A–C).

Posterior tibial artery recanalization was attempted subintimally with a 0.018-inch guide wire (V18; Boston Scientific), followed by no reentry at the common plantar artery.

After antegrade failure, percutaneous retrograde access was considered at the level of common plantar artery. Pharmacological support was used to avoid spasm; we injected 9 ml of diluted verapamil (5 mg/2 ml, diluted to 10 ml with saline solution) intra-arterially as close as possible to the foot. Local anesthesia was provided at the puncture site, close to the target vessel, and together with lidocaine was injected with 1 ml of the diluted verapamil in the subcutaneous tissue.

Retrograde access was achieved with a 21 gauge needle under fluoroscopic guidance with contrast medium injection and maximum magnification. After common plantar artery sticks, a 0.018-inch guide wire was deployed (V18), and a sheathless retrograde recanalization of the posterior tibial artery was performed, followed by rendezvous and engaging the antegrade BER II type catheter (Fig. 2D,E).

The procedure was completed by antegrade angioplasty and hemostasis of the retrograde access with a 0.014-inch wire (Pilot 200, Abbott Vascular) and a long balloon (3 × 220 mm, Bantam Alpha, Clearstream) (Fig. 2F).

In order to obtain complete revascularization, the pedal-plantar loop technique was performed through the lateral plantar artery and plantar arch, followed by retrograde recanalization of the pedal and anterior tibial arteries (Fig. 3A–C) with a 0.014-inch wire (Pilot 200) and a long balloon (3 × 220 mm, Bantam Alpha, Clearstream). Angioplasty was performed in an antegrade way.

The final angiographic result revealed complete recanalization of the limb, with patentcy of all tibial and foot arteries (Fig. 3D,F).

In the days that followed, an increase in $TcPO_2$ to 56 mm Hg was observed. The patient underwent negative-pressure wound therapy and reconstructive orthopedic and surgical therapy by positioning internal–external fixation plus an autologous skin graft (Fig. 4A).

During the clinical follow-up, the patient had good hemodynamic compensation of the foot ($TcPO_2$ of >50 mm Hg) and had good wound healing after 9 months (Fig. 4B–E).

Discussion

Depending on the stage of disease, several operative and nonoperative treatment options exist for diabetic patients with Charcot foot deformity. In the early stages, the most effective treatment is total contact cast application. In the

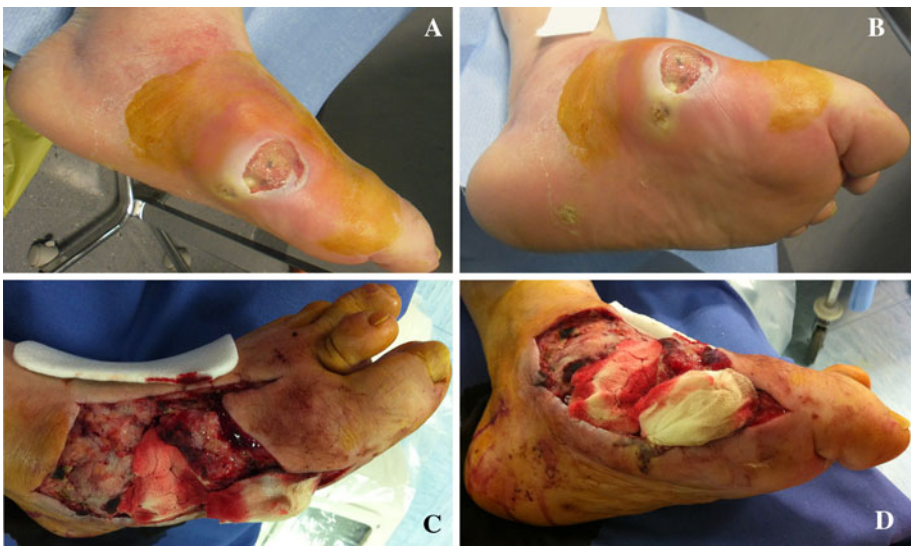

Fig. 1 Advanced stage of Charcot foot. **A, B** At admission, plantar phlegmon with dorsal involvement and cutaneous ulceration. **C, D** After surgery, drainage and infected bone amputation

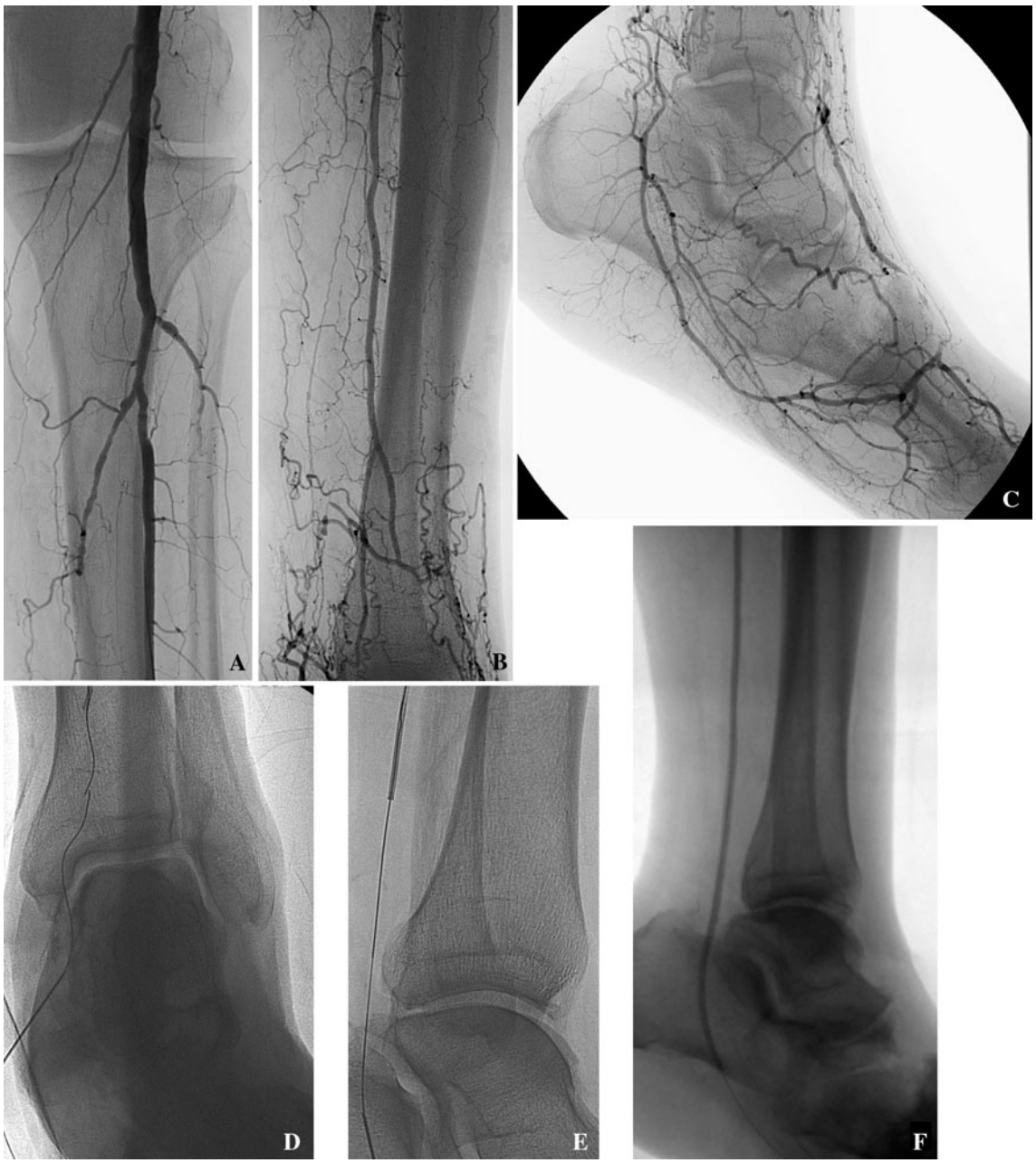

Fig. 2 **A–C** Diagnostic angiography reveals occlusion of the anterior and posterior tibial arteries, and patency of the tibioperoneal and peroneal arteries. At the foot level, pedal artery occlusion and plantar artery patency were evident. **D, E** After antegrade recanalization failure, common plantar artery retrograde access was obtained, followed by sheathless retrograde recanalization of the posterior tibial artery and rendezvous. **F** Antegrade angioplasty and hemostasis

advanced stage, surgical interventions are generally required for foot architecture reconstruction. However, recurrence of ulcer and infection is very likely after these surgical procedures, and if the lesion and infection reach the midfoot and hind foot region, a major amputation is usually required for treatment [10].

No data about the combination of Charcot foot deformity and CLI are available in the literature. However, diabetes represents an important risk factor for peripheral arterial disease and CLI [4, 8]. Infection and surgical intervention increase the requirements of blood flow for the foot to support wound healing. This fails if peripheral arterial disease is misdiagnosed or not treated.

In our opinion, in complex cases like this, revascularization therapy should be considered when CLI is present. It should be as complete as possible, with all tibial vessels recanalized, to guarantee the necessary blood flow for supporting surgical intervention, thus avoiding new infections.

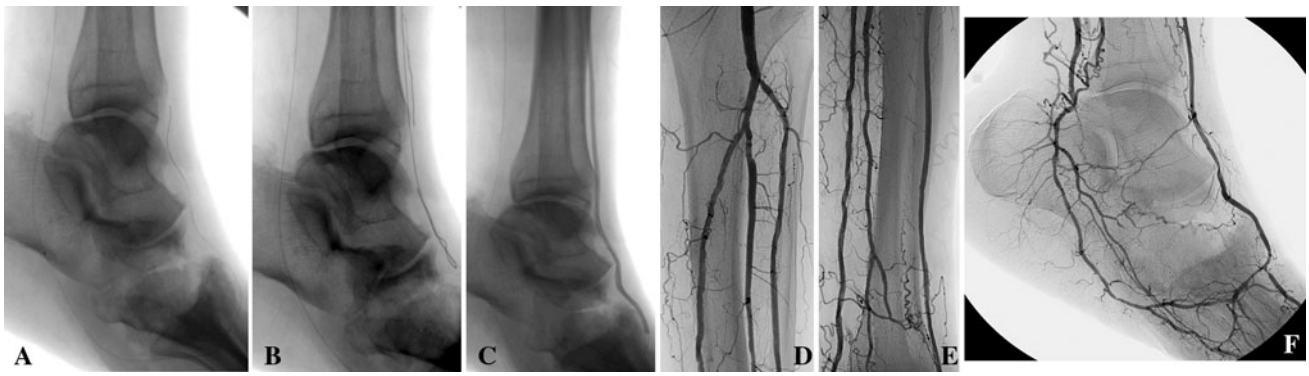

Fig. 3 **A–C** Pedal-plantar loop technique. A 0.014-inch guide wire was advanced through the lateral plantar artery and plantar arch. Retrograde recanalization of the pedal artery and anterior tibial artery was performed, followed by antegrade recanalization and angioplasty. **D–F** Angiographic control indicated patency of the all tibial and foot arteries

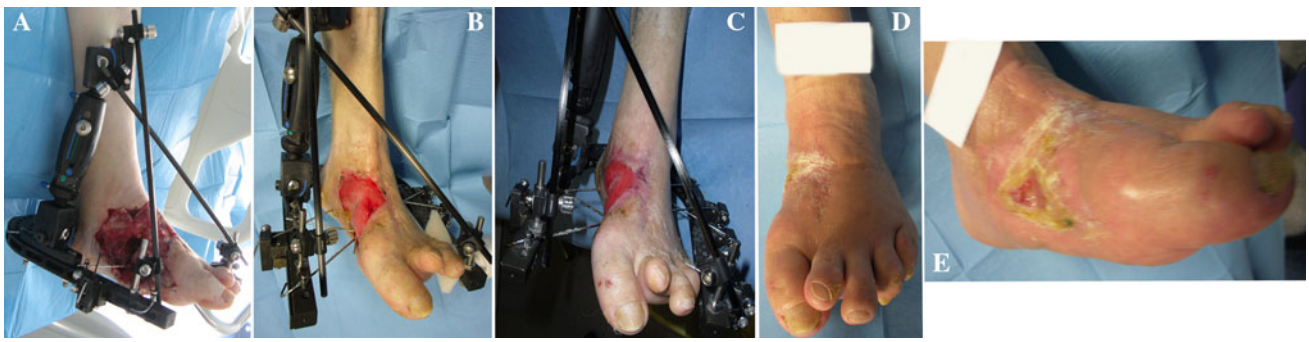

Fig. 4 **A** Surgical and orthopedic treatment, with internal–external fixation plus skin grafting. **B** At 3 months' follow-up, skin graft adhesion was evident. **C** At 6 months' follow-up, the patient showed good clinical progression of the orthopedic treatment and the skin graft, with evident graft revascularization and adhesion. **D, E** At 9 months' follow-up, after internal–external fixation removal, healing lesion with foot preservation was evident

Nevertheless, for bone and articular deformities, endovascular treatment may be very difficult as a result of the tortuosity of the occluded arteries; a combination of more and aggressive technical strategies could be required.

Here we presented a case of advanced stage Charcot foot, with bone deformity and infection, in a patient with CLI. The patient underwent endovascular recanalization, surgical and orthopedic treatment, and skin graft, with limb salvage and wound healing. We conclude that patients with Charcot foot deformity can be affected by CLI and that revascularization therapy is necessary to support surgical and orthopedic treatment, thus avoiding amputation and leading to limb and foot salvage.

Conflict of interest The authors declare that they have no conflict of interest.

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